EKLWVTVYYGVPVWKEATTT

QUERY	EKLWVTVYYGVPVWKEATTT
CONSENSUS_A	-n----------d-e--
A.GB.MA246	GN----------D-E--
A.GB.MC108	DN----------D-E--
A.KE.K89	-N----------N-E--
A.KE.Q23-CXC-CG	-N----------RD-D--
A.NG.NG1935	-N------C------D-K--
A.RW.KIG93	GN----------D-E--
A.RW.SF1703	S----------D-E--
A.SE.SE6594	D----------D-E--
A.SE.SE7253	DN----------D-E--
A.SE.SE7535	GN----------D-E--
A.SE.SE8131	-N----------D-E--
A.SE.SE8538	D----------D-E--
A.SE.SE8891	-N--------I---RD-E--
A.UG.92UG037	-N----------I--D-N--
A.UG.U455	.Q----------D-V--
A.UG.UG273A	KN----------RD-E--
A.UG.UG275A	-D----------RD-E--
CONSENSUS_B	-q----------
B.AU.MBC18	NNF----------R-----
B.AU.MBC200	-H----------R-----
B.AU.MBC925	D----------
B.AU.MBCC54	-P----------R-----
B.AU.MBCC98	D----------R--A--
B.AU.MBCD36	D-W----------R-----
B.BE.SIMI84	-Q----------
B.CN.RL42	-N----------
B.DE.D31	----------
B.DE.HAN	GN----------
B.ES.89SP061	-N----------
B.FR.HXB2	----------
B.FR.PHI120	-N----------
B.FR.PHI133	-Q----------
B.FR.PHI146	-R----------S--
B.FR.PHI153	----------
B.FR.PHI159	----------
B.FR.PIH155	----------
B.FR.PIH160	-Q----------
B.FR.PIH309	----------D--
B.FR.PIH373	----------D--
B.FR.PIH374	-Q----------
B.GA.OYI	-N----------
B.GB.AC-46	D-M----------
B.GB.CAM1	D----------
B.GB.GB8.C1	----------
B.GB.JB	-Q----------
B.GB.M23470	-N----------N-----
B.GB.M26864	-Q----------
B.GB.M30156	DQM----------
B.GB.M737677	-Q----------
B.GB.M737685	-Q----------
B.GB.MANC	----------
B.GB.MB314	GN----------
B.GB.WB	-Q----------
B.JP.ETR	-Q----------
B.JP.JH32	-Q----------A--
B.NL.3202A21	-Q----------
B.NL.68A	----------
B.NL.ENVVA	-Q----------T---
B.NL.ENVVF	-Q----------
B.NL.ENVVG	-Q----------N--
B.NL.H0320-2A12	-Q----------
B.TH.TH936705	-N----------N--
B.TT.QZ4589	-Q----------
B.TW.LM49	-N----------
B.US.85WCIPR54	-Q----------
B.US.92US657.1	-N----------D--
B.US.ADA	-N----------
B.US.ALA1	----------
B.US.BC	GN----------
B.US.BRVA	D----------N--
B.US.C26-12.1BH	GQ----------
B.US.CDC452	AN----------
B.US.DH123	-Q----------N--
B.US.ENVUS-R2	----------
B.US.JRCSF	----------T---
B.US.JRFL	----------
B.US.M02-3.SW	----------
B.US.MNCG	----------
B.US.NC7	----------
B.US.NL43E9	----------T---
B.US.NY5CG	-Q----------
B.US.P896	--T---I-------R-----
B.US.RF	-D----------
B.US.SC	-Q----------
B.US.SC141	-Q-$---------$------
B.US.SC14C	-Q----------
B.US.SF128A	------I----------
B.US.SF2	----------
B.US.SFMHS1	-Q----------T---
B.US.SFMHS11	-E----------
B.US.SFMHS16	GQ----------
B.US.SFMHS17	-Q----------
B.US.SFMHS18	-N----------
B.US.SFMHS19	-Q----------A--
B.US.SFMHS2	GQ----------
B.US.SFMHS20	----------
B.US.SFMHS21	-Q----------
B.US.SFMHS3	----------
B.US.SFMHS4	--M----------
B.US.SFMHS5	----------
B.US.SFMHS6	----------
B.US.SFMHS7	----------N--
B.US.SFMHS8	D----------
B.US.SFMHS9	DQ----------
B.US.US1	.----------
B.US.US2	GQ----------
B.US.US3	-NS----------
B.US.US4	TV----------
B.US.WC001	-QW----------
B.US.WEAU160	-N----------
B.US.WMJ22	-Q----------
B.US.WR27	-Q----------
B.US.YU2	-Q----------
CONSENSUS_C	gn----------k--
C.BI.BU910112	GN----------
C.BI.BU910213	GNS----------Q--K--
C.BI.BU910316	GN----------RD-K--
C.BI.BU910423	GN----------K--
C.BI.BU910518	LWVT-YYGVP-WKEAQPIP-
C.BI.BU910611	GN-------G-------P-
C.BI.BU910717	GNM--A----------K--
C.BI.BU910812	GN----------K--
C.BR.92BR025	GN----------K--
C.BW.96BW01B03	GN----------K--
C.BW.96BW0402	...----------KA-
C.BW.96BW0502	GN----------K--
C.BW.96BW11B01	G-S----------R--KA-
C.BW.96BW1210	-NM----$---------K--
C.BW.96BW15B03	GN------D-----R--SN-
C.BW.96BW16B01	GH----------K--
C.BW.96BW17B05	GN----------R--K--
C.DJ.DJ259A	GN----------D-NPP
C.DJ.DJ373A	GN----------Q--NP-
C.ET.ETH2220	GN----------D-SP-
C.IN.21068	GN----------N--
C.IN.301904	GN----------K--
C.IN.301905	GN----------K--
C.IN.301999	GN----------K--
C.IN.94IN11246	KD----------K--
C.SN.SE364A	GN----------K--
C.SO.SM145A	GN----------R--K--
C.UG.UG268A2	GN----------
CONSENSUS_D	-?----------
D.CD.84ZR085	DN----------
D.CD.ELI	DN----------
D.CD.JY1	-D----------
D.CD.NDK	-D----------I-------
D.CD.Z2Z6	DN----------
D.SN.SE365A2	----------
D.TZ.87TZ4622	-Q----------
D.UG.92UG024D	-QS----------
D.UG.94UG1141	G-S----------
D.UG.C971-412	-Q----------
D.UG.UG266A2	DN----------D-E--
D.UG.UG269A	GE----------
D.UG.UG274A2	-Q----------
D.UG.WHO15-474	--K----------
F.BR.BZ126A	.N----------
CONSENSUS_F1	dn----------
F1.BE.VI850	DN----------
F1.BR.93BR020.1	-N----------
F1.FI.FIN9363	DD----I----------N--
F1.FR.MP411	DN----------
CONSENSUS_F2	D?----------?---

```
F2.CM.MP255          DD--------------P---
F2.CM.MP257          D-------------------

CONSENSUS_G          nn------------Ed-d-?
G.BE.DRCBL           .-------------ED-NAP
G.FI.HH8793          NN------------ED-K--
G.GA.LBV217          GN----------A-ED-D--
G.NG.92NG083         DN------------ED-D-P
G.NG.NG1928          NN------------E--D-P
G.NG.NG1929          -N------------ED-D-S
G.NG.NG1937          NN------------ED-D--
G.NG.NG1939          NN------------ED-D-P
G.SE.SE6165          NN------------ED-D--

CONSENSUS_H          gN---------------K--
H.BE.VI991           GN---------------K--
H.BE.VI997           GN---------------K--
H.CF.90CF056         QN---------------K--

CONSENSUS_J          -?------------?D-K--
J.SE.SE9173          -N------------RD-K--
J.SE.SE9280          -D-------------D-K--

CONSENSUS_K          ??----------------?-
K.CD.EQTB11C         NN------------------
K.CM.MP535           D-----------------P-
N.CM.YBF30           -QH-----------R--E--

CONSENSUS_O          kq-YA---a-----ed--PV
O.CM.ANT70C          .Q-YA---A-----ED--PV
O.CM.CM4974          P#-YA---S------D--PV
O.CM.HIV1CA9EN       KQTYA---A-----GD-APV
O.CM.MVP5180         KQ-YA---S-----E--APV
O.GA.VI686           NH-YA---A-----ED-NPV
O.GQ.193HA           KP-YA---A-----ED--PV
O.GQ.276HA           KQ-YA---A-----ED--PV
O.GQ.341HA           RQ-YA---A-----ED-NPV
O.GQ.655HA           KQ-YA---A-----ED-IPV
AC.IN.21301          GN-------------D-E--
AC.RW.92RW009        NN-------------D-E--
AC.SE.SE9488         D----------------K--
AC.ZM.ZAM174         GN---------------K--
AC.ZM.ZAM184         GN------------R--K--
AC.ZM.ZAM716-3       GN---------------K--
ACD.SE.SE8603        D-------------D-E--
AD.KE.K124A2         .N------------RD-E--
AD.SE.SE6954         GR------------RD-E--
AD.SE.SE7108         -N-------------D-E--
AD.UG.C6080-10       DN-------------D-K--
AD.UG.UG/92/035      -N-------------D-E--
ADHU.NO.NOGIL3       GN---------------K--
ADU.CD.MAL           -D------------------
AG.GA.VI191A2        .N-----F-------D-E--
AG.NG.G3             NN------------ED-D-P
AG.SE.SE7812         .N------------RD-E--
AGHU.GA.VI354        -N--I---------RD-K-S
AGJ.AU.BFP90         KNM---------A-ED-D-I
AGJ.ML.95ML84        SN------------ED-D-I
AGJ.NG.NG3670        NNM---------A-E--D-I
AGU.CD.Z321          -N-------------D-E--
AU.NG.NG3678         DN-------------D-E--
BF.BR.93BR029.4      .N------------------
CD.BI.BU910905       GNS-----------------
CRF01_AE.CF.90CF402  DN------------RD-D-I
CRF01_AE.TH.93TH253  NN------------RD-D--
CRF01_AE.TH.A01021.  DN------------RD-D--
CRF01_AE.TH.070703   DN------------RD-D--
CRF01_AE.TH.070704   DN------------RD-D--
CRF01_AE.TH.070705   DN------------RD-D--
CRF01_AE.TH.070707   DN------------RD-D--
CRF01_AE.TH.070708   DR------------RD-D--
CRF01_AE.TH.070709   DN------------RD-N--
CRF01_AE.TH.070710   DN-------------D-N--
CRF01_AE.TH.070711   NN------------RD-D--
CRF01_AE.TH.070713   DN------------RD-D--
CRF01_AE.TH.CM240    DN------------RD-D--
CRF01_AE.TH.E11429.  DN-------------D-D--
CRF01_AE.TH.KH03     DN------------RD-E--
CRF01_AE.TH.KH08     D-------------RD-D--
CRF01_AE.TH.TH022    -N------------RD-D--
CRF01_AE.TH.TH047    DN------------RD-D--
CRF01_AE.TH.TH92014  DN-------------D-D--
CRF01_AE.TH.TH92111  NN------------RD-D--
CRF02_AG.DJ.DJ258A   .EM-----------RD-K--
CRF02_AG.FR.DJ263    .-------------RN-E--
CRF02_AG.FR.DJ264    .-------------RD-E--
CRF02_AG.NG.IBNG     .Q-------------T-E--
CRF02_AG.NG.NG1921   .D------------RD-D--
CRF02_AG.NG.NG3675   .N------------R--E--
CRF03_AB.RU.KAL1532  -N------------------
CRF03_AB.RU.KAL681   -N------------------
CRF03_AB.UA.UKR9700  -N------------RD-E--
CRF04_cpx.CY.94CY03  NN------------RD-E--
CRF04_cpx.GR.97PVCH  KDM-----------RD-E-K
CRF04_cpx.GR.97PVMY  NN------------RD-E-S
DF.BE.VI961          DN---------------N--
GH.GA.VI525          NN------------ED-E-P
GU.NG.NG3670         -N-------------D-D--
U.CD.VI1126          -D---------------N--

CONSENSUS_CPZ        ?e------------rd-?p-
CPZ.CD.CPZANT        NEDY---F------RN--P-
CPZ.GA.CPZGAB        .E------------HD-DPV
CPZ.US.CPZUS         .S--A---------RDVE--
```

Study Subject ID:04RCH80
Study Subject Clone:
Study Subject HLA:A3,A29,B7,B44,Cw7
Sequence: Known reactive 20Mer0: EKLWVTVYYGVPVWKEATTT gp160(32–51)

Possible HLA

A29	A*2901,A*2902
A3	A3.1,A*0301,A*0302,A*0304
B44	B*4402,B*4403,B*4404,B*4405,B*4406,B*4407,B*4408
B7	B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(2-9)	KLWVTVYY	A3
(6-15)	TVYYGVPVWK	A3
(1-9)	EKLWVTVYY	Cw*0702
(1-8)	EKLWVTVY	Cw*0702
(2-9)	KLWVTVYY	Cw*0702

Anchor Residues Searched

A*2902	X[E]XXXXXX[Y]
A*2902	X[E]XXXXX[Y]
A*2902	X[E]XXXXXXX[Y]
A3	X[LVM]XXXXXX[KYF]
A3	X[LVM]XXXXX[KYF]
A3	X[LVM]XXXXXXX[KYF]
B44	X[E]XXXXXX[Y]
B44	X[E]XXXXX[Y]
B44	X[E]XXXXXXX[Y]
B*4402	X[E]XXXXXX[FY]
B*4402	X[E]XXXXX[FY]
B*4402	X[E]XXXXXXX[FY]
B*4403	X[E]XXXXXX[YF]
B*4403	X[E]XXXXX[YF]
B*4403	X[E]XXXXXXX[YF]
B7	X[P]XXXXXX[LF]
B7	X[P]XXXXX[LF]
B7	X[P]XXXXXXX[LF]
B*0702	X[P]XXXXXX[L]
B*0702	X[P]XXXXX[L]
B*0702	X[P]XXXXXXX[L]

B*0703	X[P]XXXXXX[L]
B*0703	X[P]XXXXX[L]
B*0703	X[P]XXXXXXX[L]
B*0705	X[P]XXXXXX[L]
B*0705	X[P]XXXXX[L]
B*0705	X[P]XXXXXXX[L]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(22–31)	RPGGKKRYKL	RPGGKKKYKL	RPGGKKKYKL	B7	
p24(174–184)	AEQASQDVKNW	AEQASQEVKNW	AEQASQEVKNW	B*4402	
p24(174–184)	AEQASQDVKNW	AEQASQEVKNW	AEQASQEVKNW	B*4402,B44	
p24(223–231)	GPSHKARVL	GPGHKARVL	GPGHKARVL	B7	
gp160(31–40)	AENLWVTVYY	TEKLWVTVYY	AEQLWVTVYY	B*4402	
gp160(31–40)	AENLWVTVYY	TEKLWVTVYY	AEQLWVTVYY	B44	
gp160(208–217)	VSFEPIPIHY	ISFEPIPIHY	VSFEPIPIHY	A29	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*07	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*0702	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7?	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
gp160(376–384)	PNCGGEFFY	FNCGGEFFY	FNCGGEFFY	A29	
gp160(419–427)	RIKQIINMW	KIKQIINMW	RIKQIINMW	A29,A32	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A*0301	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A3	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B*0702	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B7	
Nef(77–85)	RPMTYKAAL	RPMTYKAAV	RPMTYKAAV	B*0702	
Nef(175–184)	DPEKEVLQWK	DPEREVLEWR	DPEKEVLVWK	B7	
Nef(190–198)	AFHHVAREK	AFHHVAREL	AFHHMAREL	A3	

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(22–31)	Gag(22–31)	RPGGKKRYKL	HIV-1 infection	human(B7)	[Jin (2000)]
• This B7 epitope is one of three subdominant CTL responses detected in a long-term non-progressor • A dominant B7 epitope was de£ned using conventional methods, and three additional sub-dominant HLA B7 epitopes were de£ned by £rst using a non-anchor based strategy, EpiMatrix, to identify 2078 possible epitopes in the autologous HIV-1, followed by B7 anchor residue prediction to narrow the set to 55 peptides for experimental testing					

Table 2: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(174–184)	p24(306–316 LAI)	AEQASQDVKNW		human(B*4402)	[Brander & Goulder(2001)]
• C. Brander notes this is a B*4402 epitope					
p24(174–184)	p24(306–316 LAI)	AEQASQDVKNW		human(B*4402,B44)	[Brander & Walker(1997)]
• Pers. Comm. from D. Lewinsohn to C. Brander and B. Walker, C Brander *et al*., this database, 1999					
p24(223–231)	p24()	GPSHKARVL	HIV-1 infection	human(B7)	[Goulder (2000)]
• The CTL-dominant response was focused on this epitope in a HIV+ Caucasian living in Boston – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa				

Table 3: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(31–40)	gp160(30–39 WEAU)	AENLWVTVYY	HIV-1 infection	human(B*4402)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*4402 epitope				
gp160(31–40)	gp160(30–39 WEAU)	AENLWVTVYY	HIV-1 infection	human(B44)	[Borrow (1997), Goulder (1997a), Borrow & Shaw(1998)]
	• Two CTL lines from the patient WEAU were studied – one had an optimal peptide of (A)AENLWVTVYY, and the other (A)AENLWVTVY, and both responded equally well with one or two N-term Alanines • Rapidly post-infection, a strong immunodominant response was observed against this epitope • The naturally occurring forms of the peptide found in WEAU were tested as targets for early WEAU CTLs – the form TENLWVTVY was as reactive as the wild type AENLWVTVY – but the forms AKNLWVTVY, AGNLWVTVY, AANLWVTVY did not serve as targets • The glutamic acid in the second position is a B44 anchor residue • [Goulder (1997a)] and [Borrow & Shaw(1998)] are reviews of immune escape that summarizes this study in the context of CTL escape to £xation				
gp160(208–217)	gp120()	VSFEPIPIHY	HIV-1 exposed seronegative	human(A29)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women				
gp160(298–307)	gp120(298–307)	RPNNNTRKSI	HIV-1 infection	human(B*07)	[Ferris (1999), Hammond (1995)]
	• The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains an N-linked glycosylation site that is glycosylated in Env • Peptide that had been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) (RPNDNTRKSI) was recognized a 100-fold more ef£ciently than either glycosylated or non-glycosylated RPNNNTRKSI • Position 5 is not involved with HLA B*07 binding, so is probably important for TCR recognition • HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively				
gp160(298–307)	gp120(302–312 HXB2)	RPNNNTRKSI	HIV-1 infection	human(B*0702)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*0702 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(298–307)	gp120(302–312 HXB2)	RPNNNTRKSI	HIV-1 infection	human(B7)	[Safrit (1994)]
	• CTL from two acute seroconversion cases				
gp160(298–307)	gp120(303–312 IIIB)	RPNNNTRKSI	HIV-1 infection	human(B7?)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RPNNNTRKDI and RPNNNTRKGI, naturally occurring variants, were found in non-transmitting mother – ability to recognize these variants has not yet been determined				
gp160(298–307)	gp120(302–311 Clade B)	RPNNNTRKSI	HIV-1 infection	human(B7)	[Wilson (1998)]
	• The extent of CTL interclade cross-reactivity from CTL isolated from individuals newly infected with B clade virus was studied, and extensive cross-reactivity was observed • Two HLA B7 individuals had CTL response to B_LAI, A_92UG037 and C_92BR025 gp160, but were B clade strain MN non-responders – the authors note that the B7 epitope RPNNNTRKSI is immunodominant, conserved between the LAI and clade A and C strains, but is very divergent in MN (RPNYNKRKRI), and that this epitope might be dominating the speci£city of the response in the HLA B7 individuals				
gp160(376–384)	gp120(376–384 IIIB)	PNCGGEFFY	HIV-1 infection	human(A29)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • PNCRGEFFY was an escape variant				
gp160(419–427)	gp120(419–427)	RIKQIINMW?	HIV-1 infection	human(A29,A32)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was A29 and responded to RIKQIINMW, and another responder was A32 and these are thought to be presenting molecules • The sequence is unclear – Betts calls both peptide 30 and peptide 32 gp120 419–427 and the peptide sequences are not provided				
gp160(770–780)	gp41(768–778 NL43)	RLRDLLLIVTR	HIV-1 infection	human(A*0301)	[Takahashi (1991)]
	• CD8+ T cell clone				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(770–780)	gp41(768–778 NL43)	RLRDLLLIVTR	HIV-1 infection	human(A3)	[Cao (1997)]
	• The consensus peptide of clade B is RLRDLLLIVTR • The consensus peptide of clades A, C and E is RLRDFILIVTR and it is less reactive • The consensus peptide of clade D is SLRDLLLIVTR and it is less reactive				
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B*0702)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*0702 epitope				
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B7)	[Brander & Walker(1995)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study				

Table 4: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Bauer (1997)]
	• Structural constraints on the Nef protein may prevent escape • Noted in Brander 1999, this database, to be B*0702				
Nef(175–184)	Nef(175–184)	DPEKEVLQWK	HIV-1 infection	human(B7)	[Jin (2000)]
	• This a B7 epitope, a subdominant CTL response, was de£ned by an un-conventional approach used to predict epitopes in an HLA B7+ long-term non-progressor • Three additional sub-dominant HLA B7 epitopes were de£ned using EpiMatrix, a non-anchor based strategy for de£ning potential epitopes, which highlighted 2078 possible epitopes in the autologous HIV-1 derived from the study subject, followed by B7 anchor residue prediction which narrowed the set to 55 peptides, three of which could serve as functional CTL epitopes				
Nef(190–198)	Nef(190–198 LAI)	AFHHVAREK	HIV-1 infection	human(A3)	[Hadida (1995)]
	• Naturally occurring L to K anchor substitution abrogates A2 binding, but permits HLA-A3 binding				

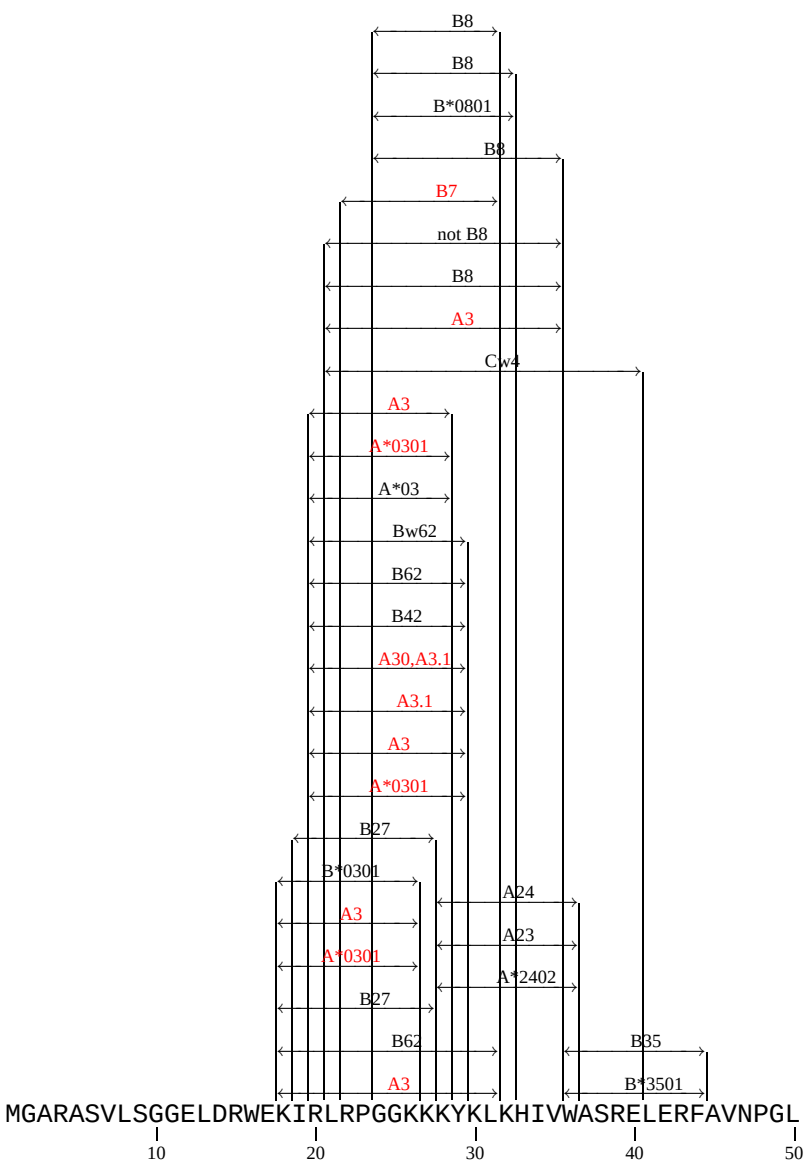
Table 5: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(33–42)	gp120(32–41 LAI)	KLWVTVYYGV	MN rec gp160	human(A2)	[Dupuis (1995)]
	• CTL from HLA-A2 positive subject react with this peptide				
gp160(33–42)	Env(32–41 Clade B)	KLWVTVYYGV	HIV-1 infection plus HIV-1 MN rgp160 stimulation	human(A2.1)	[Kundu (1998)]
	• Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and £fty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identi£ed in gp160, of which 25 had a high or intermediate binding af£nity • Eleven peptides were studied that had high HLA-A2 binding af£nity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses				
gp160(36–46)	gp120()	VTVYYGVPVWK	HIV-1 infection	human(A11 and A*6801)	[Threlkeld (1997)]
	• Study of the £ne speci£city of an A3-like-HLA-super-type epitope (the A3-super-type includes A*0301, A*1101, A*3101, A*3301, and A*6801) • The A3 super-type is characterized as a hydrophobic or hydroxyl containing anchor residue at position 2, and a positive charge in the C-term position • While most lines were speci£c, a promiscuous cloned CTL line was derived from an HIV+ donor that could recognize this epitope presented by either A11 or A*6801				
gp160(37–46)	gp120(37–46 LAI)	TVYYGVPVWK	gp160 vaccinia vaccine	human(A*0301)	[Johnson (1994b)]
	• Multiple CTL clones obtained from two vaccinees • C. Brander notes that this is an A*0301 epitope in the 1999 database				
gp160(37–46)	gp120(37–46 LAI)	TVYYGVPVWK	gp160 vaccinia vaccine	human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0301 epitope				
gp160(37–46)	Env()	TVYYGVPVWK	DNA multi-epitope vaccine	SJL/J HLA trans-genic mice(A11)	[Ishioka (1999)]
	• A minigene vaccine construct encoding 6 HLA 2.1 and 3 HLA A11 restricted CTL epitopes, the universal Th cell epitope PADRE (pan-DR epitope) and an ER translocating signal sequence was constructed • The epitopes were chosen for dominant recognition by CTLs during HBV and HIV infections in humans • HLA transgenic mice were used for quantitating *in vivo* immunogenicity of DNA vaccines encoding HLA-restricted CTL epitopes – strong				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(37–46)	gp120(37–46)	TVYYGVPVWK	Live recombinant canarypox (CP) virus vaccine containing multiple HIV-1 genes (HIV-1 MN gp120, HIV-1 LAI gp41, HIV-1 LAI Gag, HIV-1 LAI protease)	human(A3)	[Carruth (1999)]
	• CD4+ and CD8+ Gag and Env speci£c CTL responses were detected in only 1/5 vaccinated volunteers, and were not detectable 1 year after vaccination • CTL responses to epitopes SLYNTVATL and TVYYGVPVWK from HIV+ control patients were used as positive controls • The study explored why vaccinees were non-responsive – non-response was not due to inherent defects or differences in the ability of these individuals to process and present antigen				
gp160(37–46)	gp120(37–46 LAI)	TVYYGVPVWK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
	• Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • One had a response to this epitope, the other did not • [Goulder (1997a)] is a review of immune escape that summarizes this study				
gp160(37–46)	gp120(38–41 LAI)	TVYYGVPVWK	gp160 vaccinia vaccine	human(A3.1)	[Johnson (1994a)]
	• Highly conserved epitope recognized by multiple CTL clones from vaccinee				
gp160(37–46)	gp120(37–46 LAI)	TVYYGVPVWK	gp160 vaccinia vaccine	human(A3.1)	[Hammond (1995), Ferris (1999)]
	• This peptide can be processed for HLA-A3.1 presentation by TAP-1/2 independent and dependent pathways				
gp160(37–46)	gp120(37–46 LAI)	TVYYGVPVWK	HIV-1 infection	human(B*0301)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(38–48)	gp120(45–55)	VYYGVPVWKEA	HIV-1 infection	human(Cw7)	[Nehete (1998)]
	• Three long-term non-progressors and one asymptomatic HIV+ individual were studied and found to have HLA class I C-restricted CD8+ Env-speci£c CTLs – Cw7 speci£c CTL were found against three peptides, including this one • HLA-C antigens are expressed on lymphoid cells to a lesser extent than either HLA-A or -B • HLA-C confers protection against lysis by natural killer cells and by non-MHC-restricted effector T cells and Cw7 directly governs this resistance to lysis – the authors hypothesize that pathogens that inhibit antigen expression and class I expression may particularly down regulate Cw7, thus triggering non-MHC restricted killing			
gp160(42–51)	gp120(42–51 PV22)	VPVWKEATTT	HIV-1 infection	human(B*5501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*5501 epitope				
gp160(42–51)	gp120(42–51 PV22)	VPVWKEATTT	HIV-1 infection	human(B55)	[Brander & Walker(1995)]
	• P. Johnson, unpublished				

p17 CTL Map


B8
B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
MGARASVLSGGELDRWEKIRLRPGGKKKYKLKHIVWASRELERFAVNPGL
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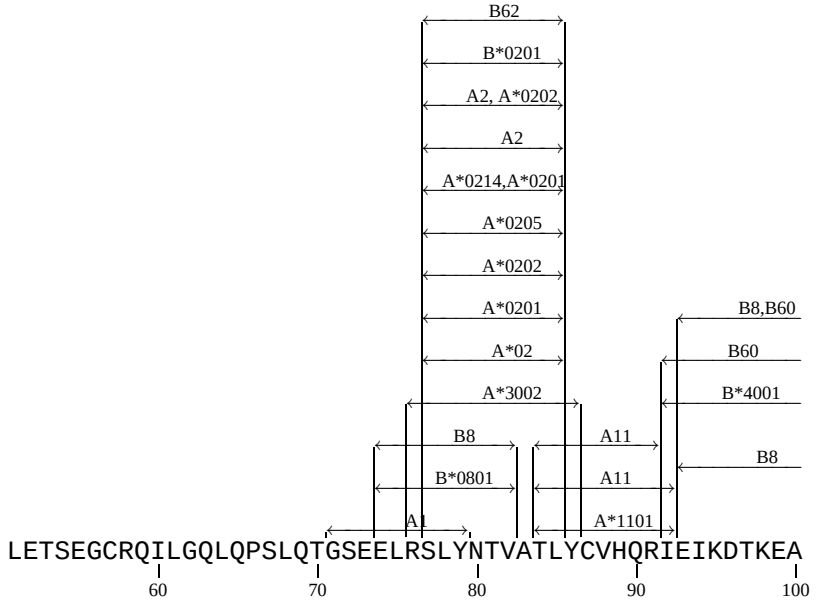
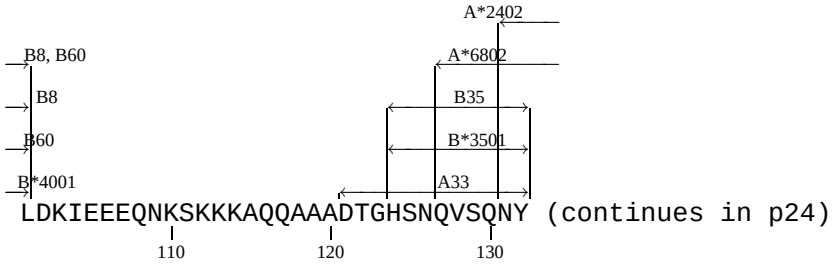
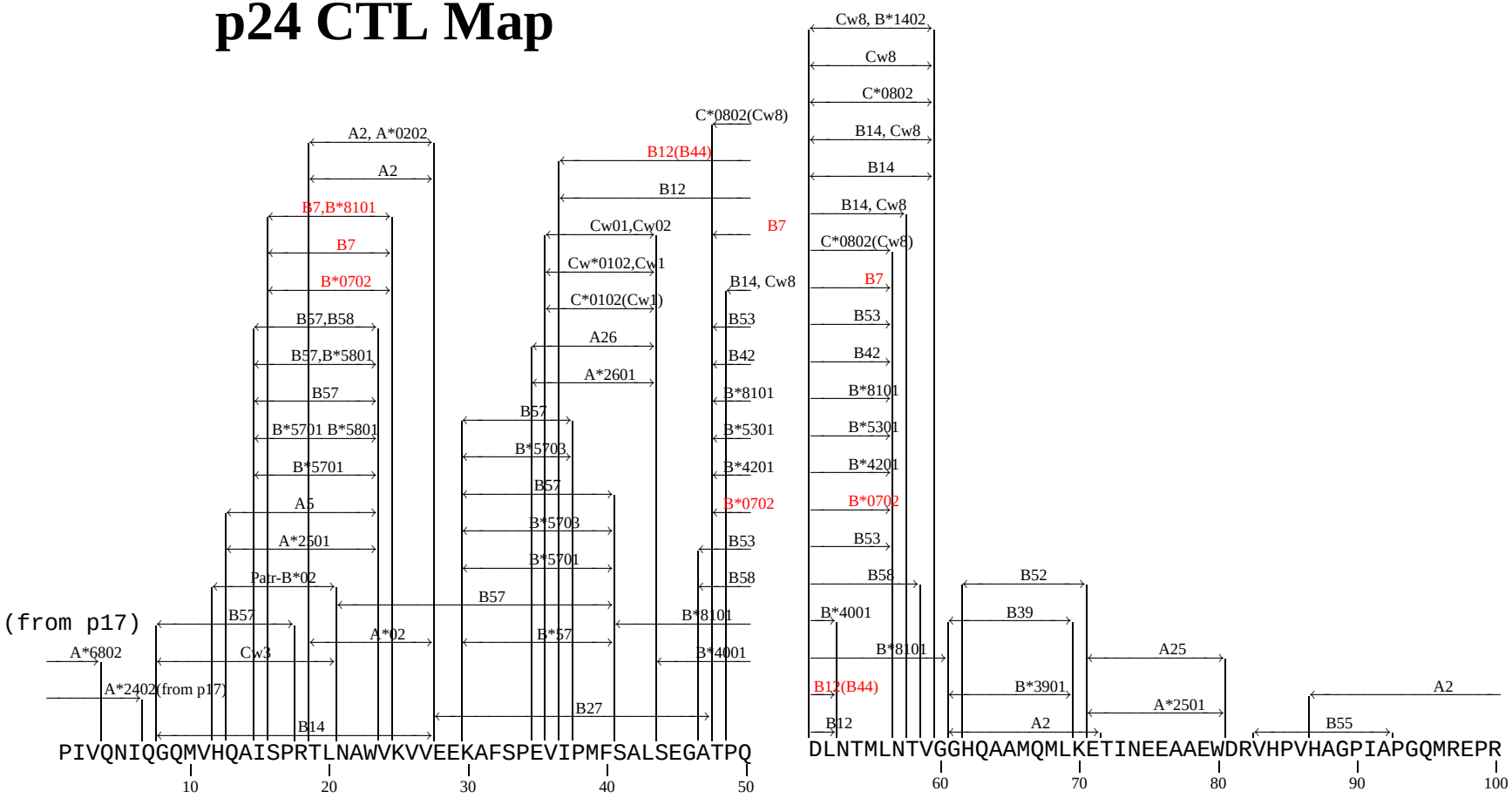
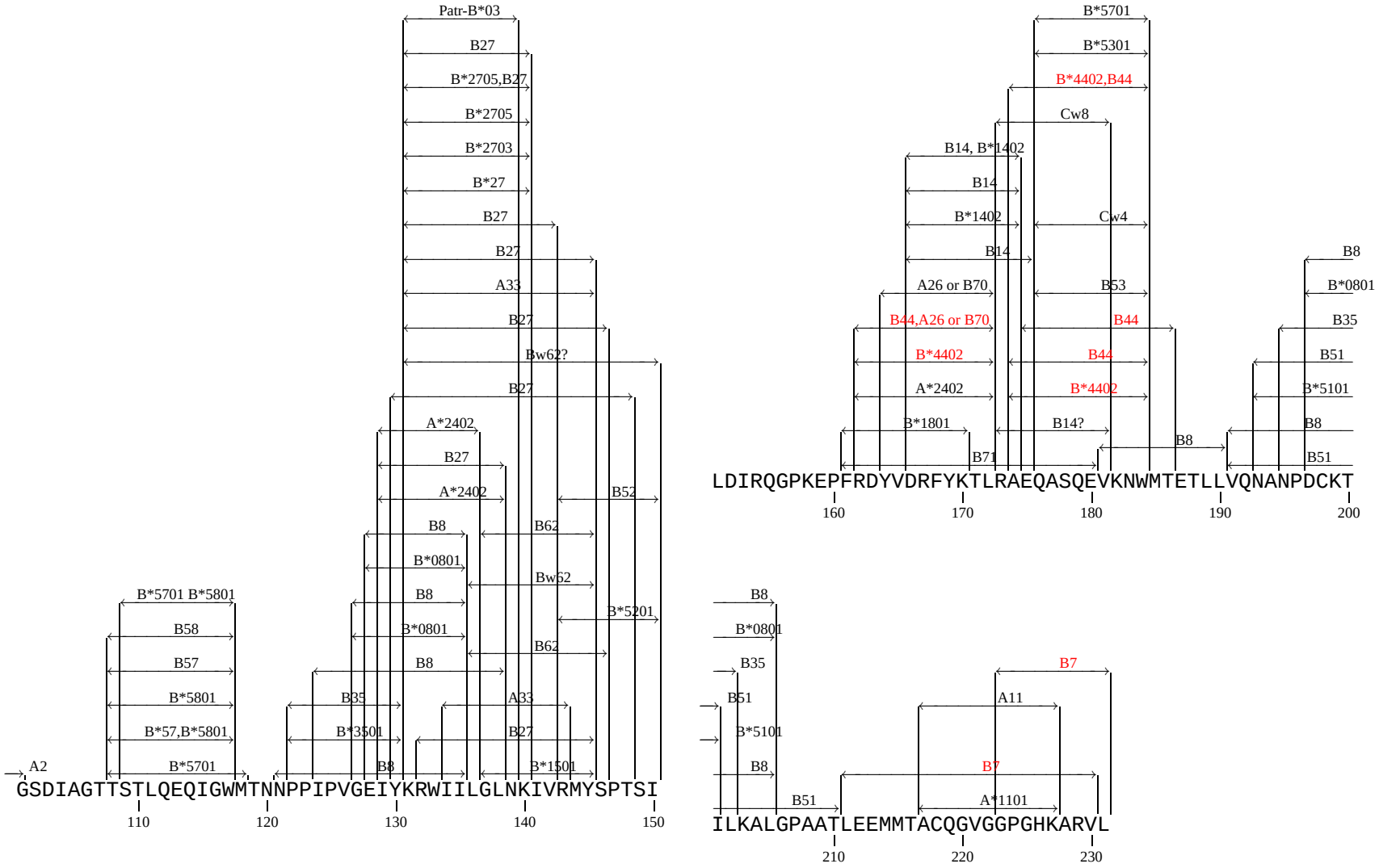
30
40
50


B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
B60
B*4001
B8
LETSEGCRQILGQLQPSLQTGSEELRSLYNTVATLYCVHQRIEIKDTKEA
60
70
80
90
100

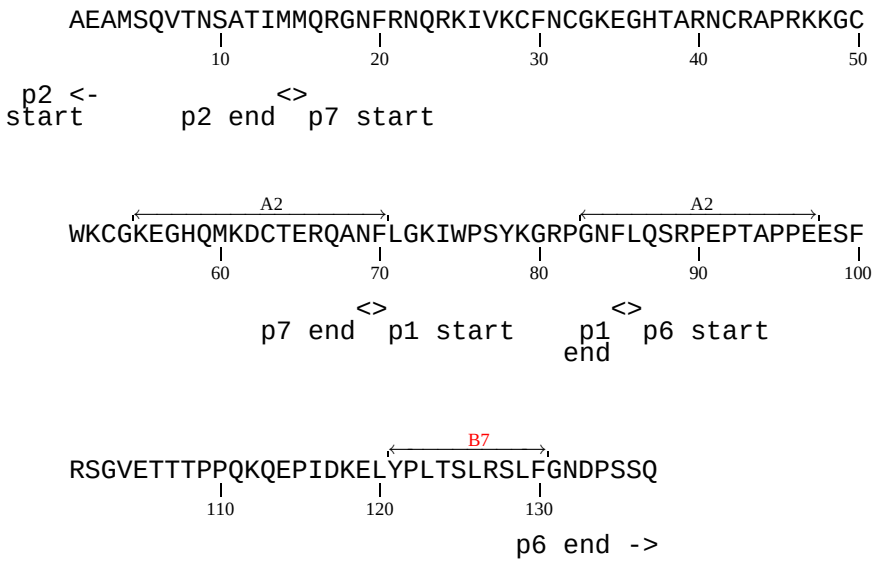

A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
LDKIEEEQNKSKKKAQQAAADTGHSNQVSQNY (continues in p24)
110
120
130

p24 CTL Map

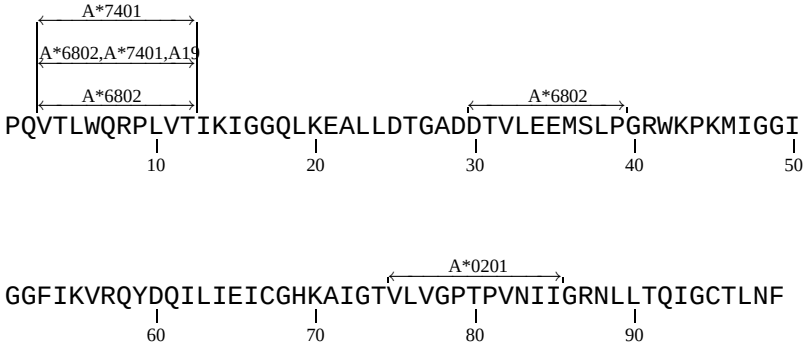

(from p17)
A*6802
A*2402
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*57
B27
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
PIVQNIQGQMVHQAISPRTLNAWVKVVEEKAFSPEVIPMFSALSEGATPQ
10
20
30
40
50
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
B12(B44)
B12
B52
B39
A25
B*3901
A*2501
A2
A2
B55
DLNTMLNTVGGHQAAMQMLKETINEEAAEWDRVHPVHAGPIAPGQMREPR
60
70
80
90
100


Patr-B*03
B27
B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
B*0801
B62
B57
B8
B*5801
B35
A33
B*57,B*5801
B*3501
B27
A2
B*5701
B8
B*1501
GSDIAGTTSTLQEQIGWMTNNPPIPVGEIYKRWIILGLNKIVRMYSPTSI
110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
B8
A26 or B70
B53
B*0801
B44,A26 or B70
B44
B35
B*4402
B44
B51
A*2402
B*4402
B*5101
B*1801
B14?
B8
B8
B71
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B8
B*0801
B35
B51
B*5101
B8
B51
B7
A11
B7
A*1101
ILKALGPAATLEEMMTACQGVGGPGHKARVL
210
220
230

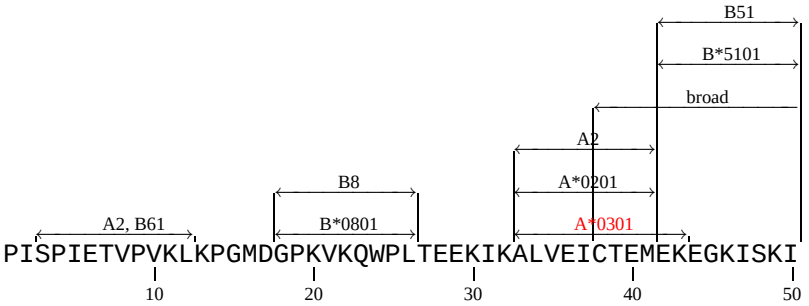
p2p7p1p6 CTL Map

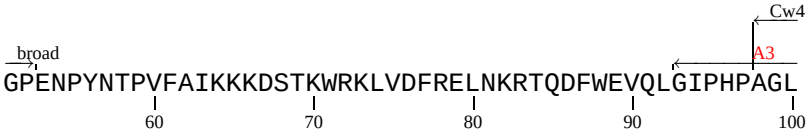


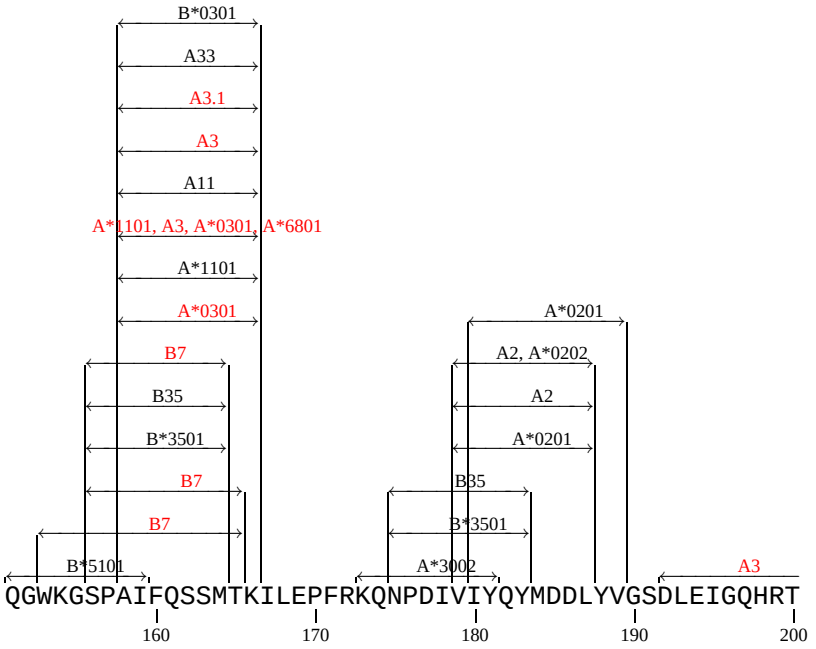
Protease CTL Map

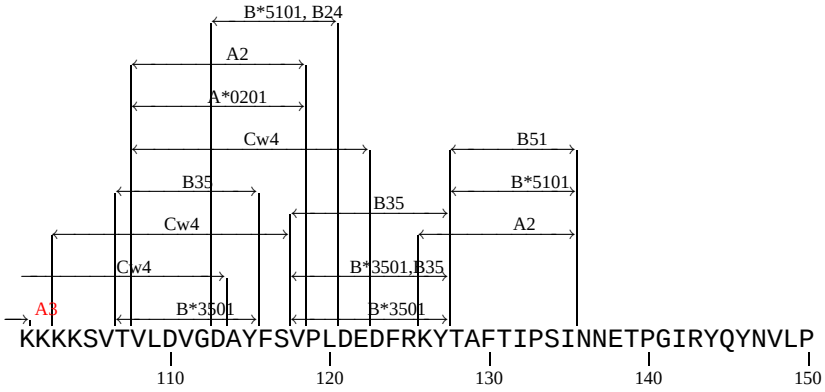


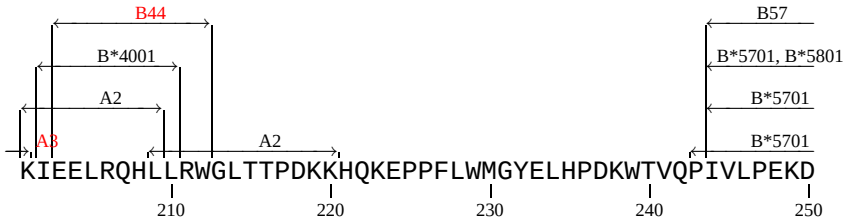
RT CTL Map


B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50


Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200


B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150


B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
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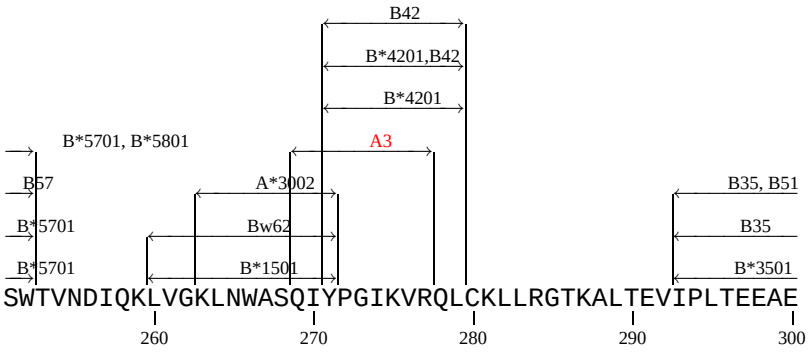
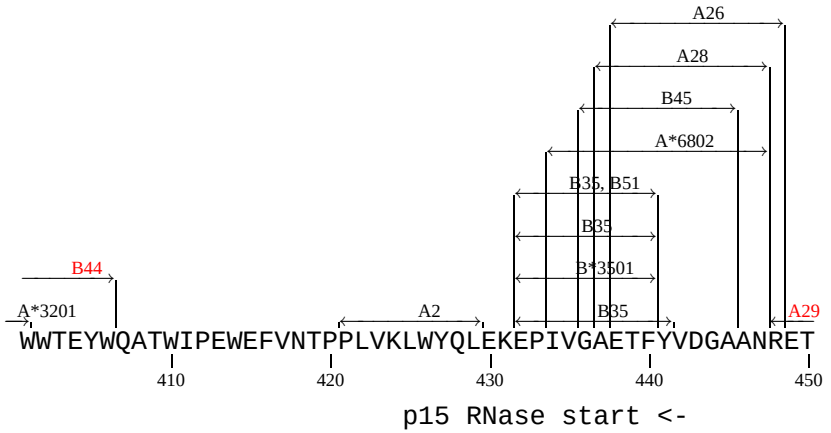
230
240
250





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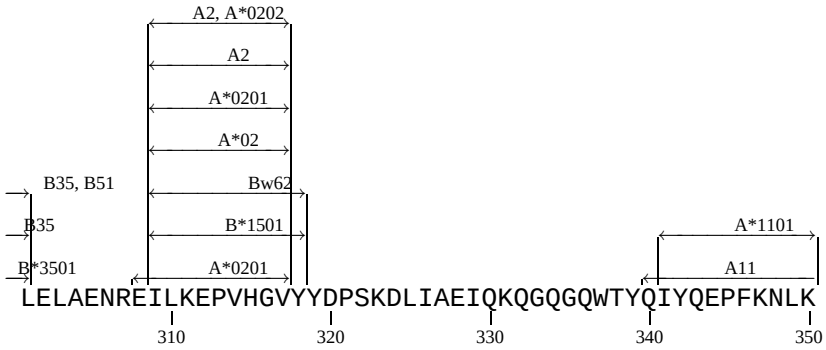
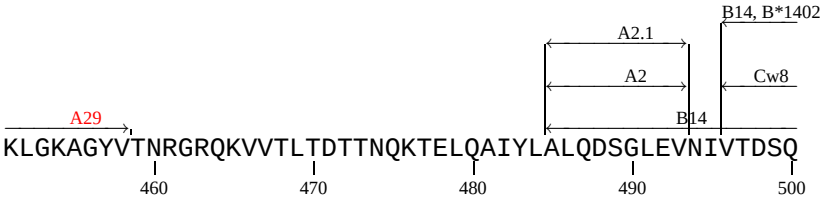
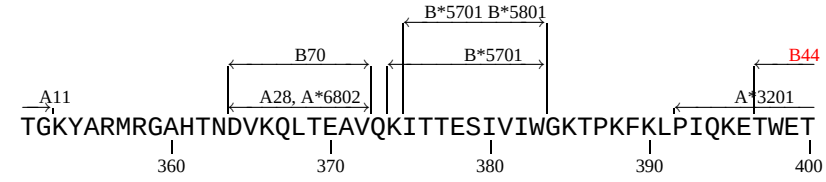
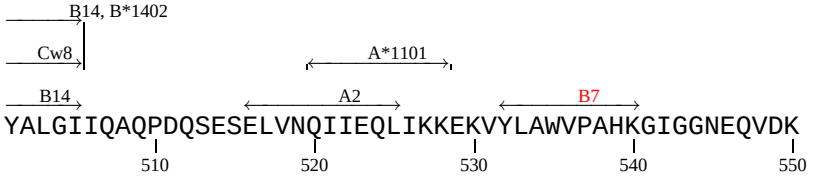




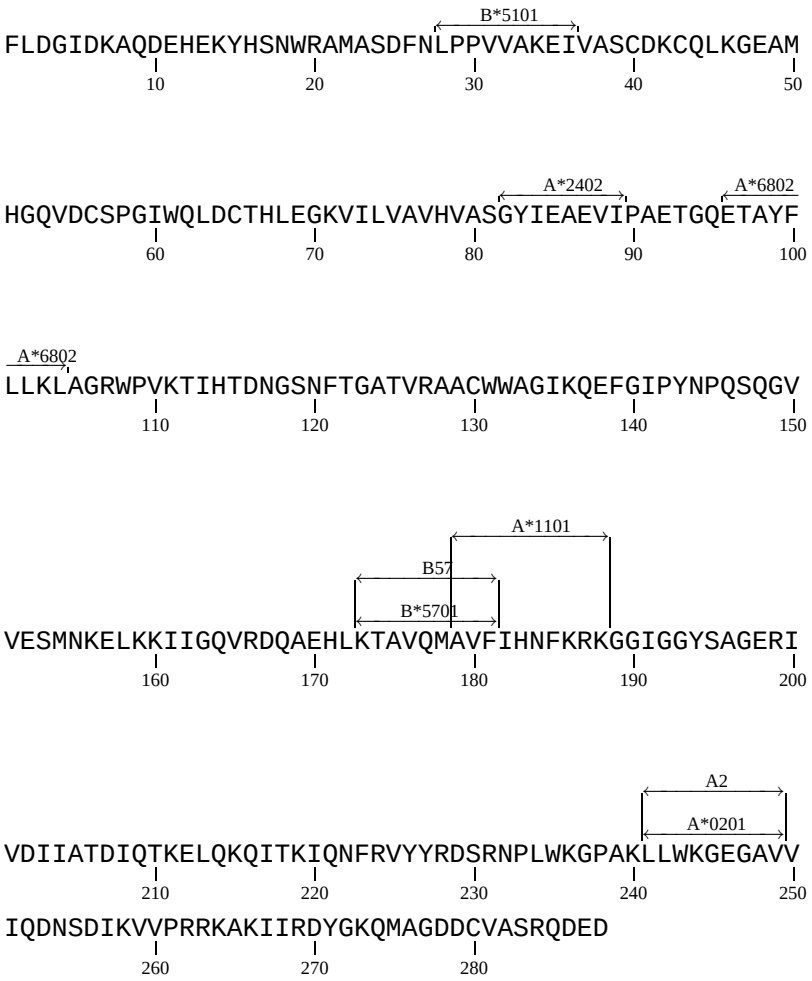




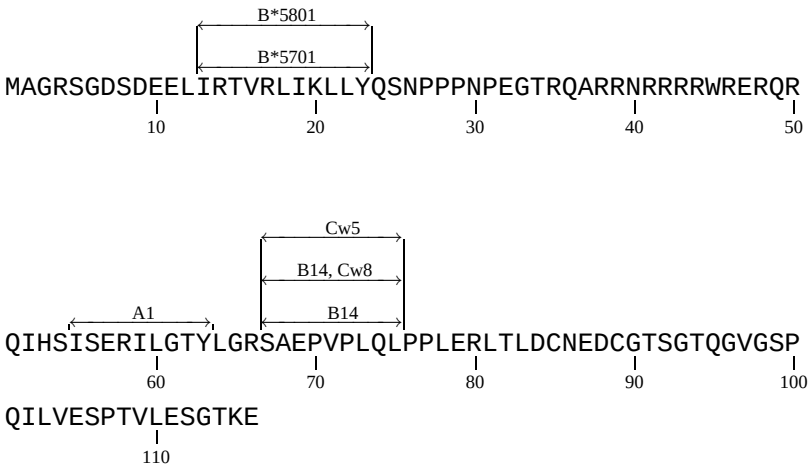
LVSAGIRKVL

-> p15 RNase end

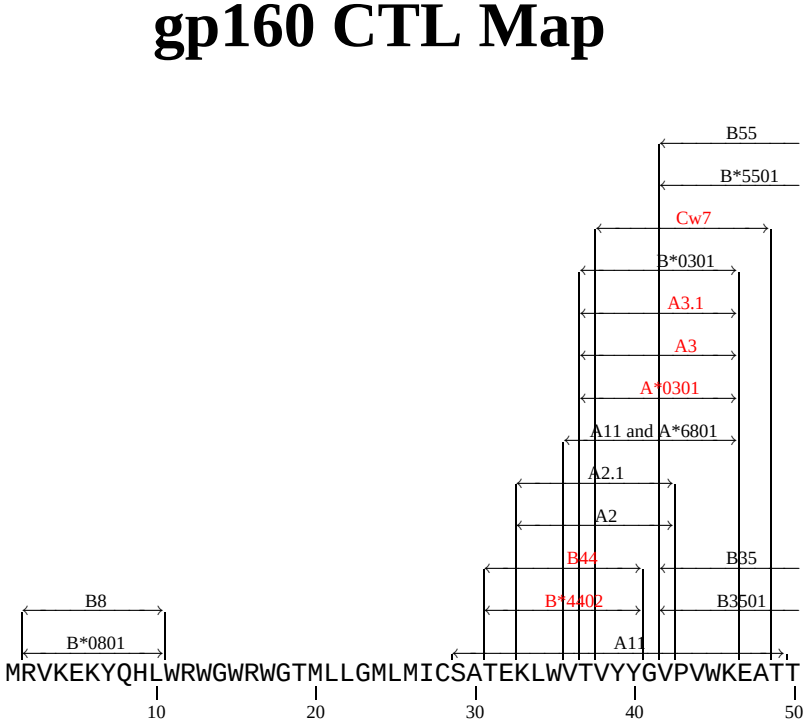
Integrase CTL Map



Rev CTL Map



gp160 CTL Map



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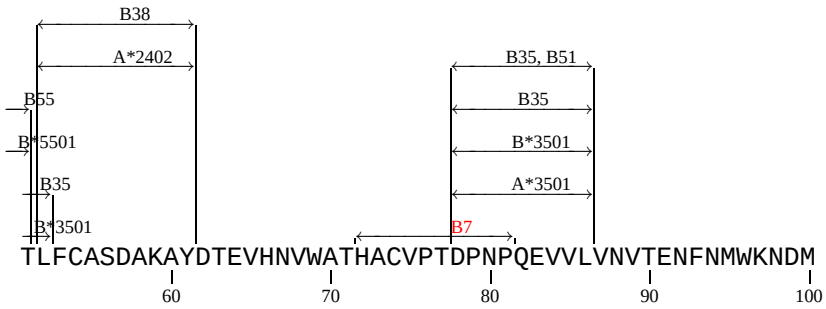
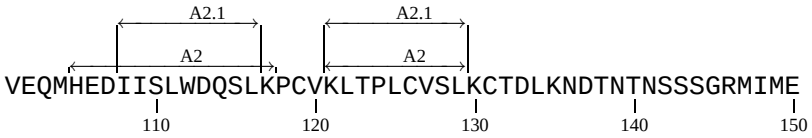
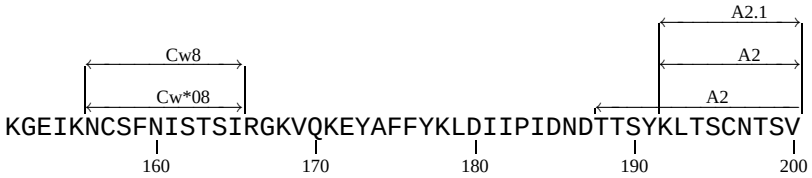
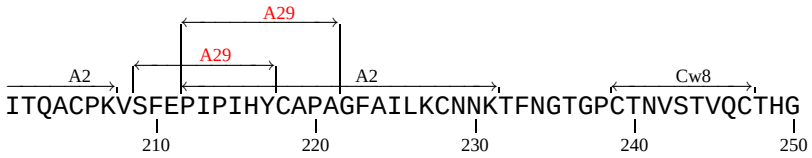
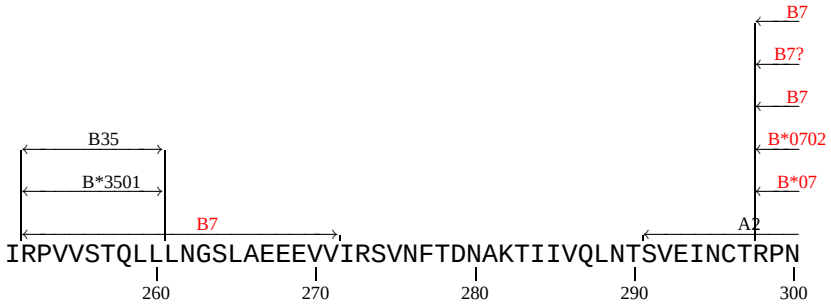
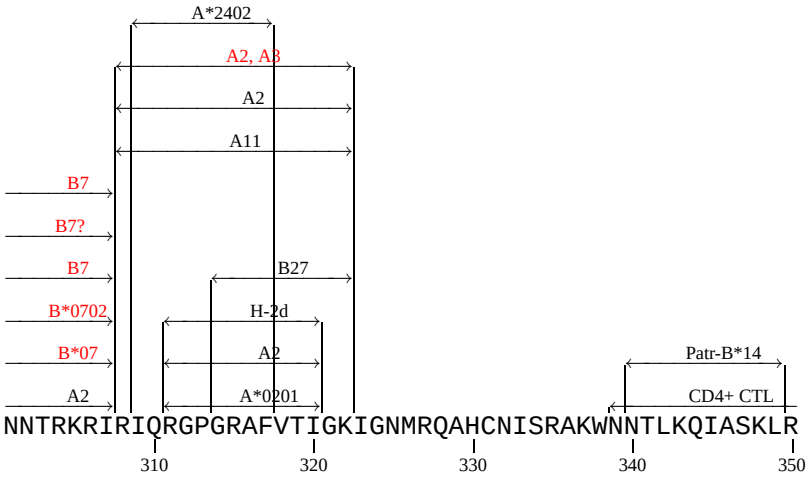


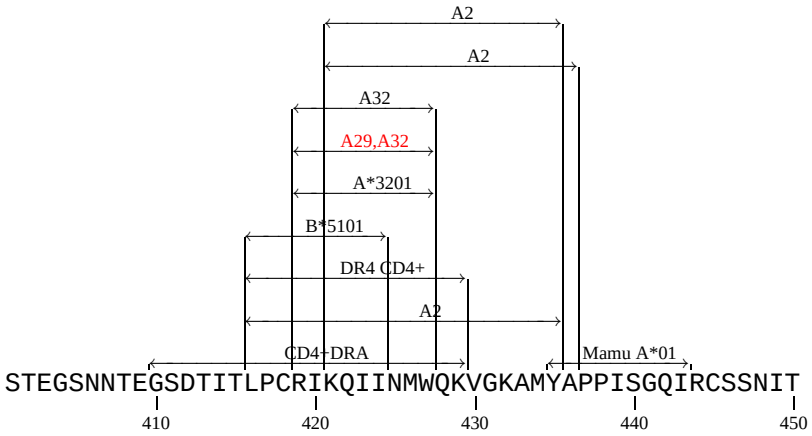


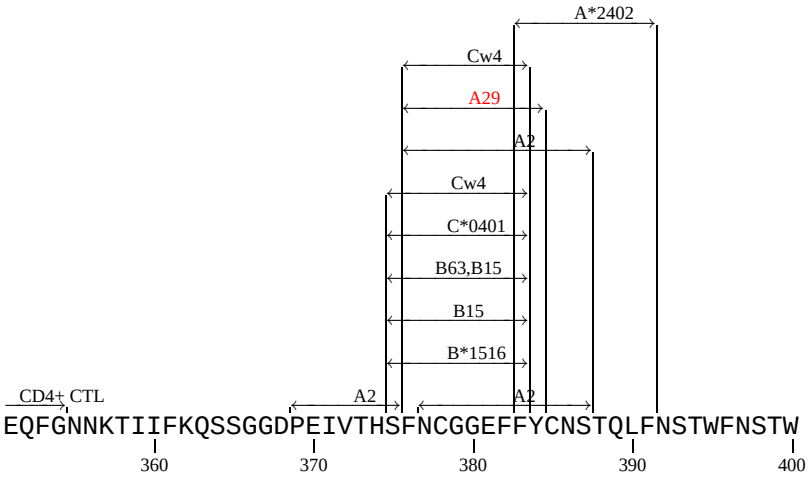




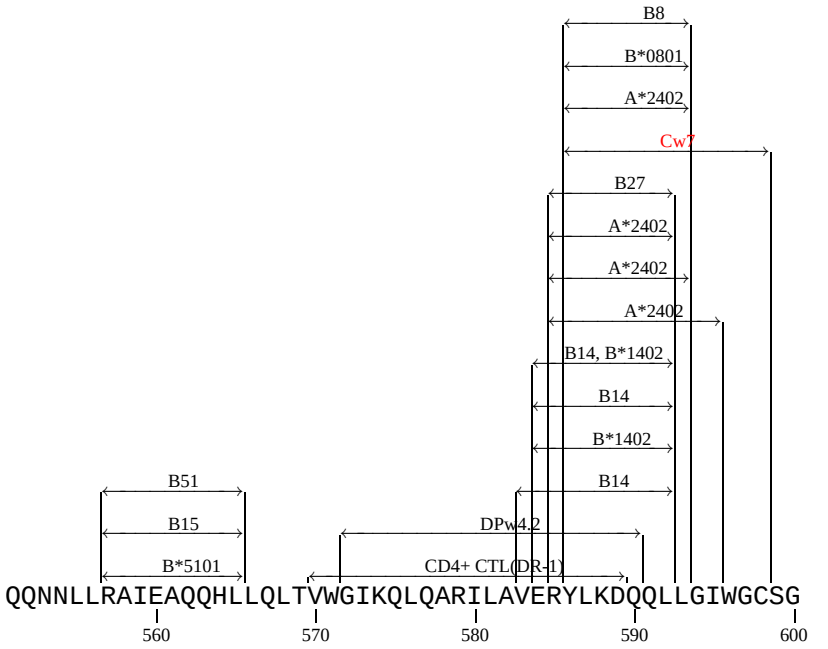


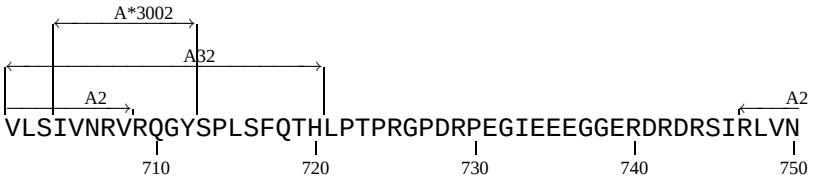


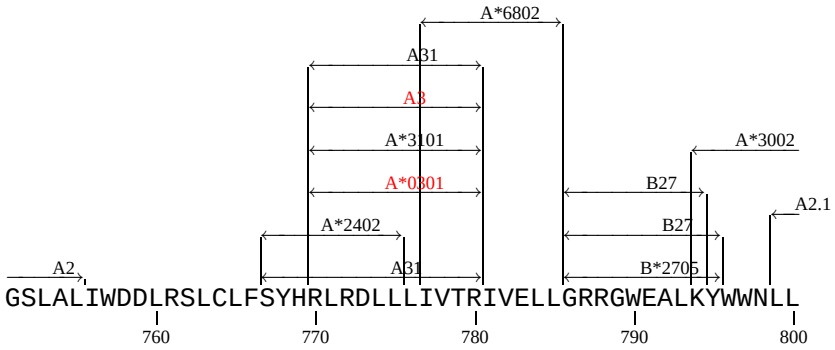




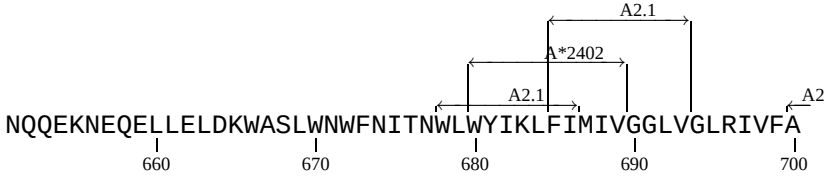
gp120 end <> gp41 start


B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600


A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A2
A31
A*3002
B27
B27
B*2705
A2.1
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650


A2.1
A*2402
A2.1
A2
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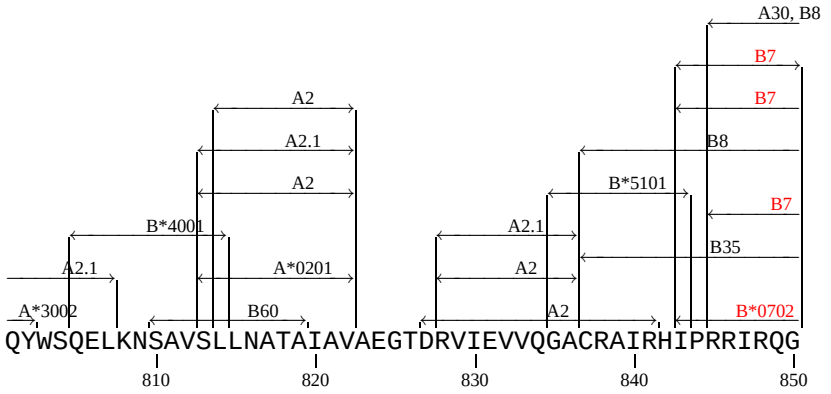
660
670
680
690
700


A30, B8
B7
B7
A2
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A*0201
A2
A*3002
B60
A2
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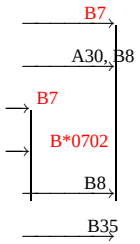
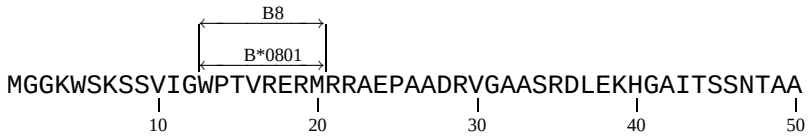
840
850

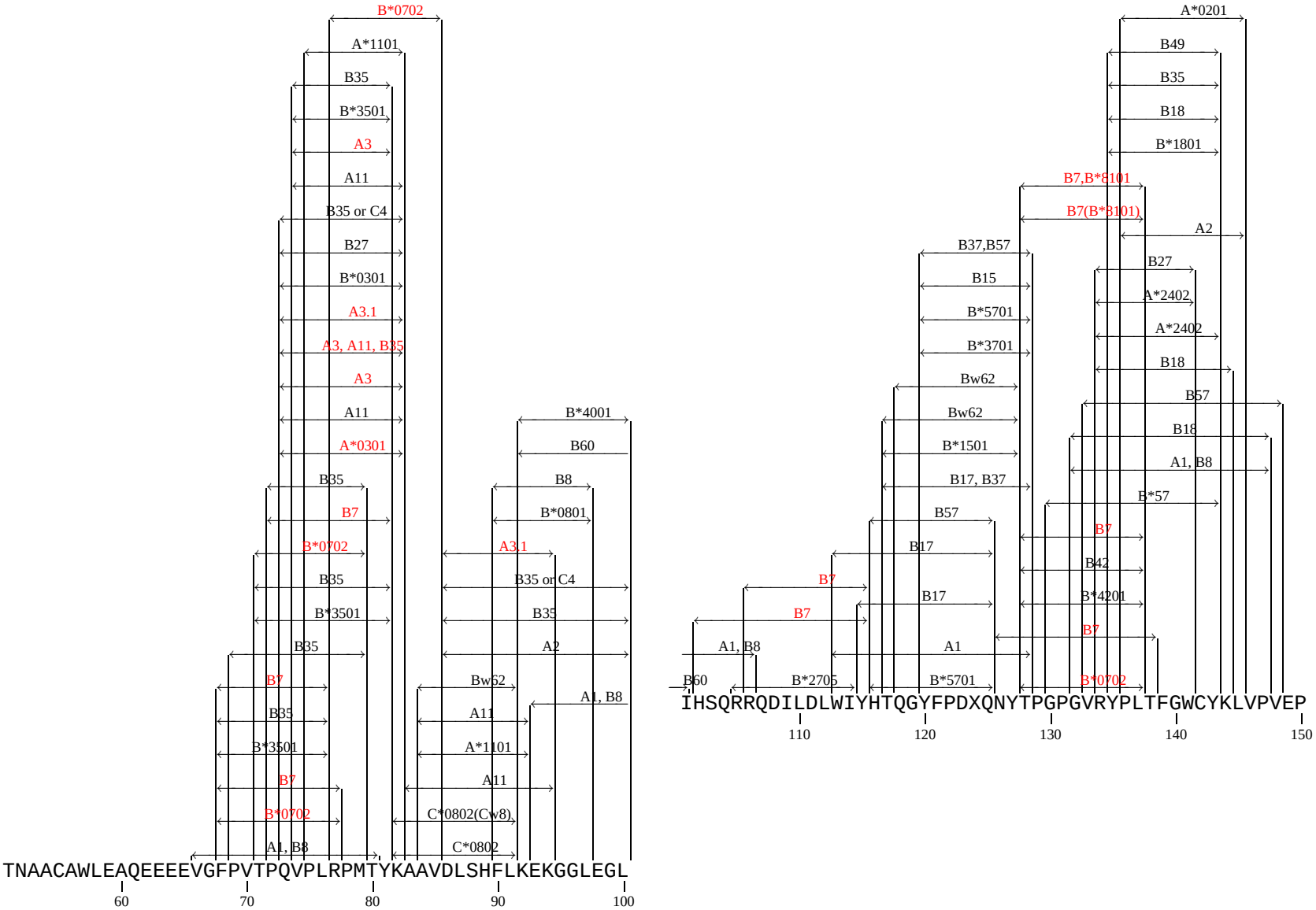


LERILL

-> gp41 end

Nef CTL Map




B*0702
A*1101
B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
B35
B*3501
B7
B*0702
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A1, B8
A11
A*1101
A11
C*0802(Cw8)
C*0802
TNAACAWLEAQEEEEVGFPVTPQVPLRPMTYKAAVDLSHFLKEKGGLEGL
60
70
80
90
100
A*0201
B49
B35
B18
B*1801
B7,B*8101
B7(B*8101)
A2
B37,B57
B27
B15
A*2402
B*5701
A*2402
B*3701
B18
Bw62
B57
Bw62
B18
B*1501
A1, B8
B17, B37
B*57
B57
B7
B17
B42
B7
B17
B*4201
B7
B7
A1, B8
A1
B60
B*2705
B*5701
B*0702
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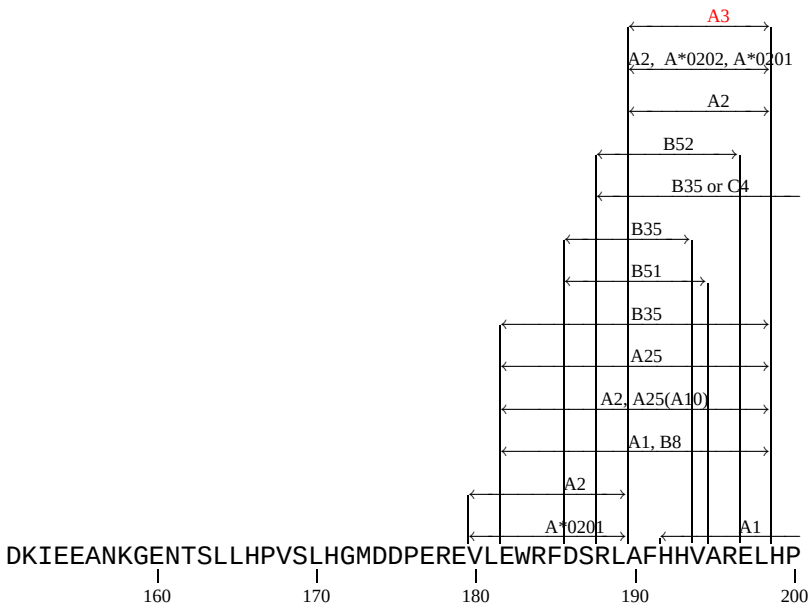
120
130
140
150



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[Brander & Walker(1997)] C. Brander & B. Walker. Systematic identi£cation of optimal HIV-1 CTL epitopes. *HIV Molecular Immunology Database* pages IV–1 to IV–11, 1997.

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Retroviruses **10, Supp 2**:S73–S75, 1994a. (Medline: 95169519) Notes: Volunteers were immunized with recombinant vaccinia virus expressing HIV-1 gp160 (vac-env) and boosted with recombinant gp160 (rgp160). CTL clones were analyzed for HLA restriction and speci£city. An immunodominant HLA-A3.1 restricted epitope was observed that showed very little sequence variation among B subtype sequences, (TVYYGVPVWK). Naturally occurring variants of this peptide were able to stimulate reactivity. Two additional CD8+ CTL epitopes from vaccinees were characterized, as well as two CD4+ CTL epitopes.

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